



Hidden hearing loss with envelope following responses (EFRs): The off-frequency problem

Encina-Llamas, Gerard; Parthasarathy, Aravindakshan; Harte, James Michael; Dau, Torsten; Kujawa, Sharon G.; Shinn-Cunningham, Barbara; Epp, Bastian

Publication date:
2017

Document Version
Publisher's PDF, also known as Version of record

[Link back to DTU Orbit](#)

Citation (APA):
Encina-Llamas, G., Parthasarathy, A., Harte, J. M., Dau, T., Kujawa, S. G., Shinn-Cunningham, B., & Epp, B. (2017). *Hidden hearing loss with envelope following responses (EFRs): The off-frequency problem*. Poster session presented at 40th MidWinter Meeting of the Association for Research in Otolaryngology, Baltimore, Maryland, United States.

General rights

Copyright and moral rights for the publications made accessible in the public portal are retained by the authors and/or other copyright owners and it is a condition of accessing publications that users recognise and abide by the legal requirements associated with these rights.

- Users may download and print one copy of any publication from the public portal for the purpose of private study or research.
- You may not further distribute the material or use it for any profit-making activity or commercial gain
- You may freely distribute the URL identifying the publication in the public portal

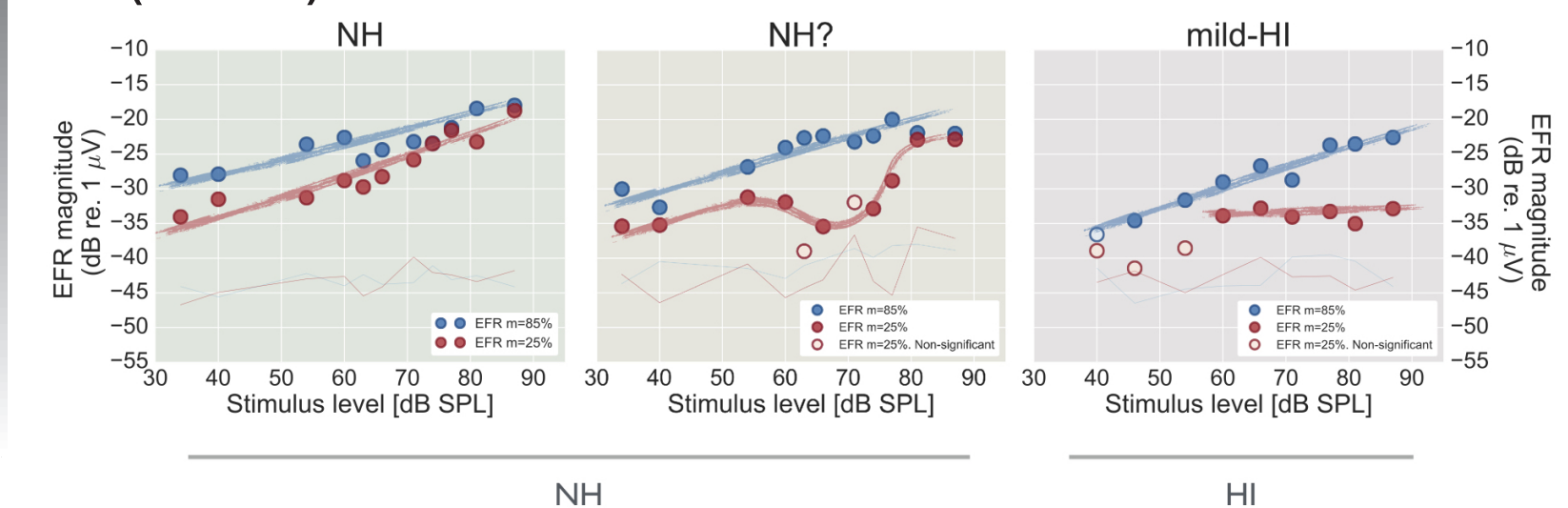
If you believe that this document breaches copyright please contact us providing details, and we will remove access to the work immediately and investigate your claim.

Introduction

Recent animal studies have shown that noise over-exposure can cause the loss of auditory nerve (AN) fiber synapses without causing hair cell loss (see Kujawa and Liberman (2015) for a review). This AN fiber synapses loss has been termed "hidden hearing loss" or "synaptopathy", since it is not reflected in the traditional pure-tone threshold. The envelope following response (EFR) has been proposed as a potential objective method to assess synaptopathy in humans (i.e., Bharadwaj *et al.*, 2015). Encina-Llamas *et al.*, (2016) reported different trends in EFR level-growth functions in normal-hearing (NH) and mild hearing-impaired (HI) listeners. The EFR is a gross encephalographic potential that represents the encoding of the envelope of the stimulus, arising from synchronized neural activity from all excited frequencies and fibers. In this study, an computational model of the AN was used to investigate the effects of off-frequency contributions (i.e. away from the characteristic place of the stimulus) and the differential loss of different AN fiber types on EFR level-growth functions.

Research Question

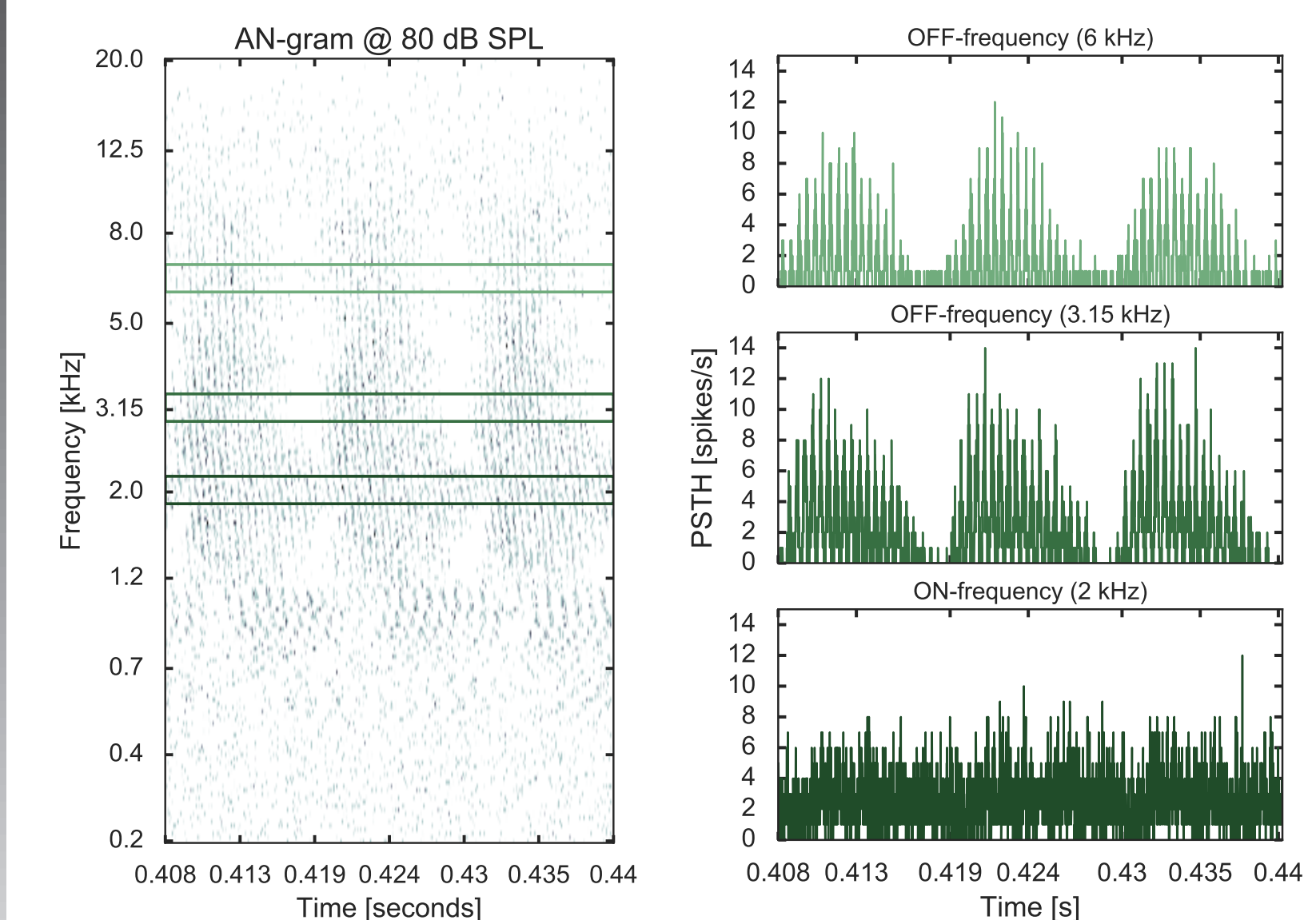
- Can a phenomenological AN computational model explain the different trends observed in the EFR level-growth functions in NH and mild-HI listeners reported in Encina-Llamas *et al.*, (2016)?



Methods

Model:

- Humanized AN model (Zilany *et al.*, 2014).
- 200 characteristic frequencies (CF), ranging from 0.2 to 20 kHz.
- Synapses per IHC are simulated by several independent computations of each AN CF (about 100 per CF). Synaptopathy is simulated by computing less of such independent computations.



Stimuli:

- $f_c = 2000$ Hz @ $f_m = 93$ Hz (as in Encina-Llamas *et al.* (2016)).

Levels:

- EFR level-growth: 5 to 100 dB SPL, 5 dB steps.
- EFR in noise: -30 to 40 dB SNR, 5 dB steps. Fix SAM at 70 dB SPL.

Modulations:

- Full $m = 85\%$, Shallow $m = 25\%$.

Simulations I

Normal-hearing:

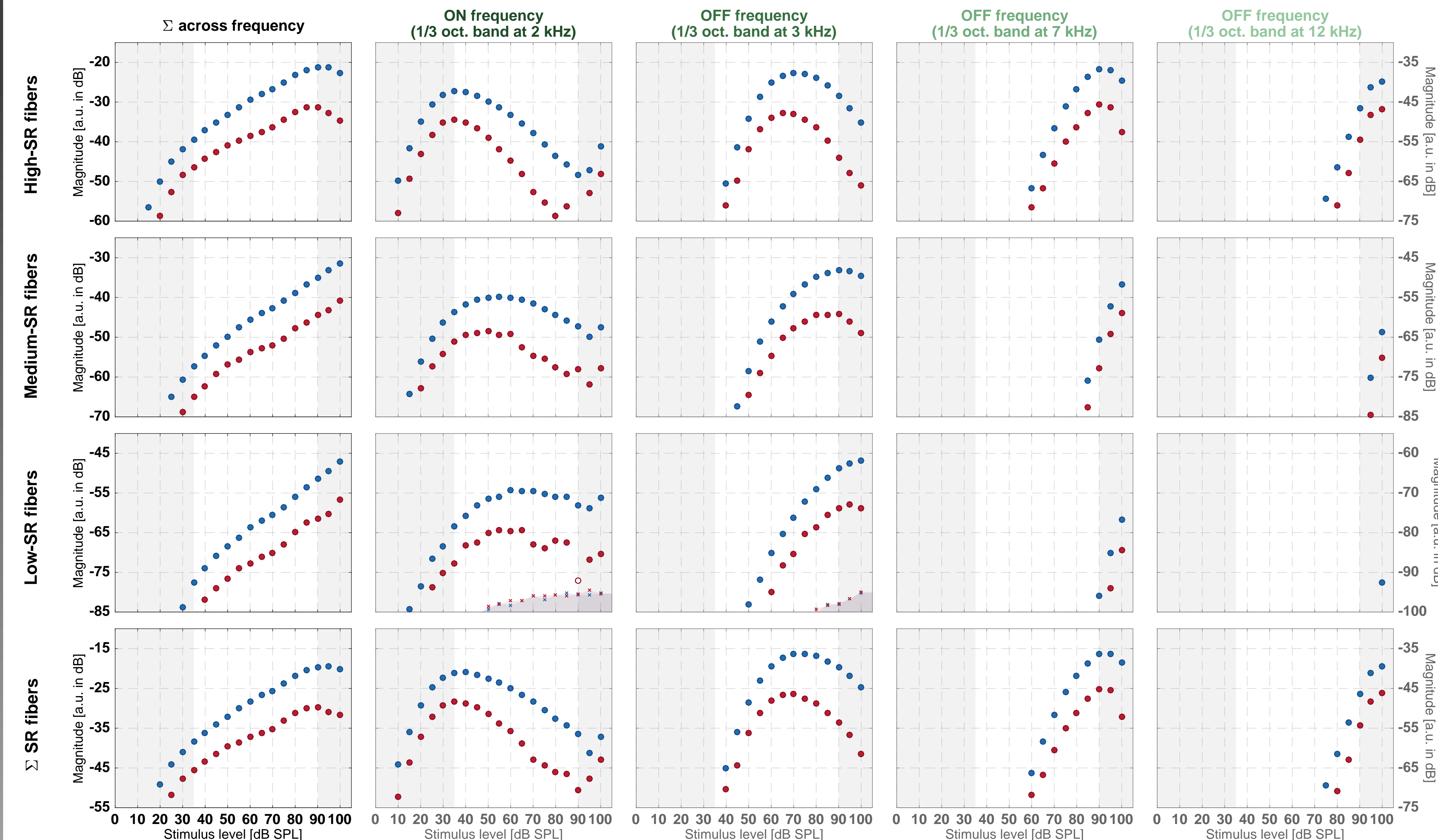


Fig.1 Simulated EFR level-growth functions using full ($m = 85\%$, blue markers) and shallow ($m = 25\%$, red markers) amplitude modulated tones. Results show different trends when summing across frequency, at on-frequency and at several off-frequency bands, and a complete dominance of the high-SR fibers in the summed response across fiber type (lower row). The grey shaded areas delimit the input level range that can be compared with the recorded EFR data in Encina-Llamas *et al.* (2016).

EFR are gross encephalographic potentials, representing the sum of all electrical neural activity at the modulation frequency. Therefore, the panel to be compared with the recorded EFR data is the lower left one.

Synaptopathy:

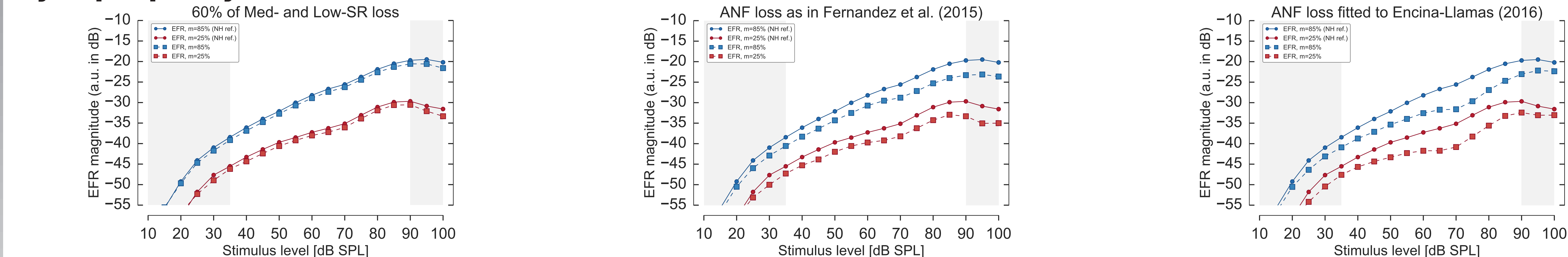


Fig.2 Simulated EFR level-growth functions with a 60% of loss of medium- and low-spontaneous rate (SR) ANF.

Fig.3 Simulated EFR level-growth functions with the same ANF loss as in Fernandez *et al.* (2015) adapted from the mice to the human cochlea.

Fig.4 Simulated EFR level-growth functions to match the response from the NH? group in Encina-Llamas *et al.* (2016).

Mild hearing-impaired:

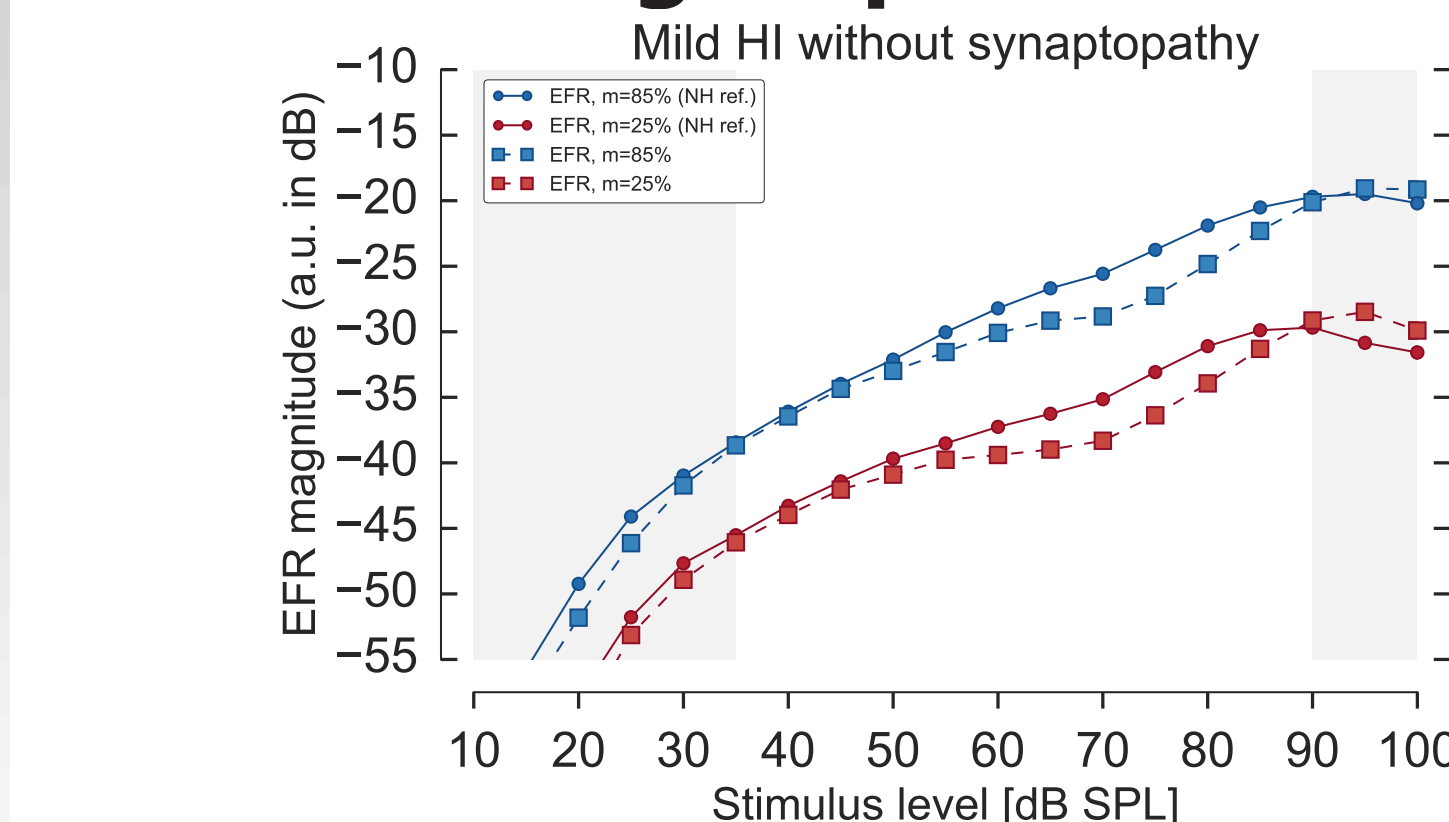


Fig.5 Simulated EFR level-growth functions for the mild-HI group in Encina-Llamas *et al.* (2016). The group averaged audiogram is fitted assuming 2/3 of OHC dysfunction and 1/3 of IHC dysfunction.

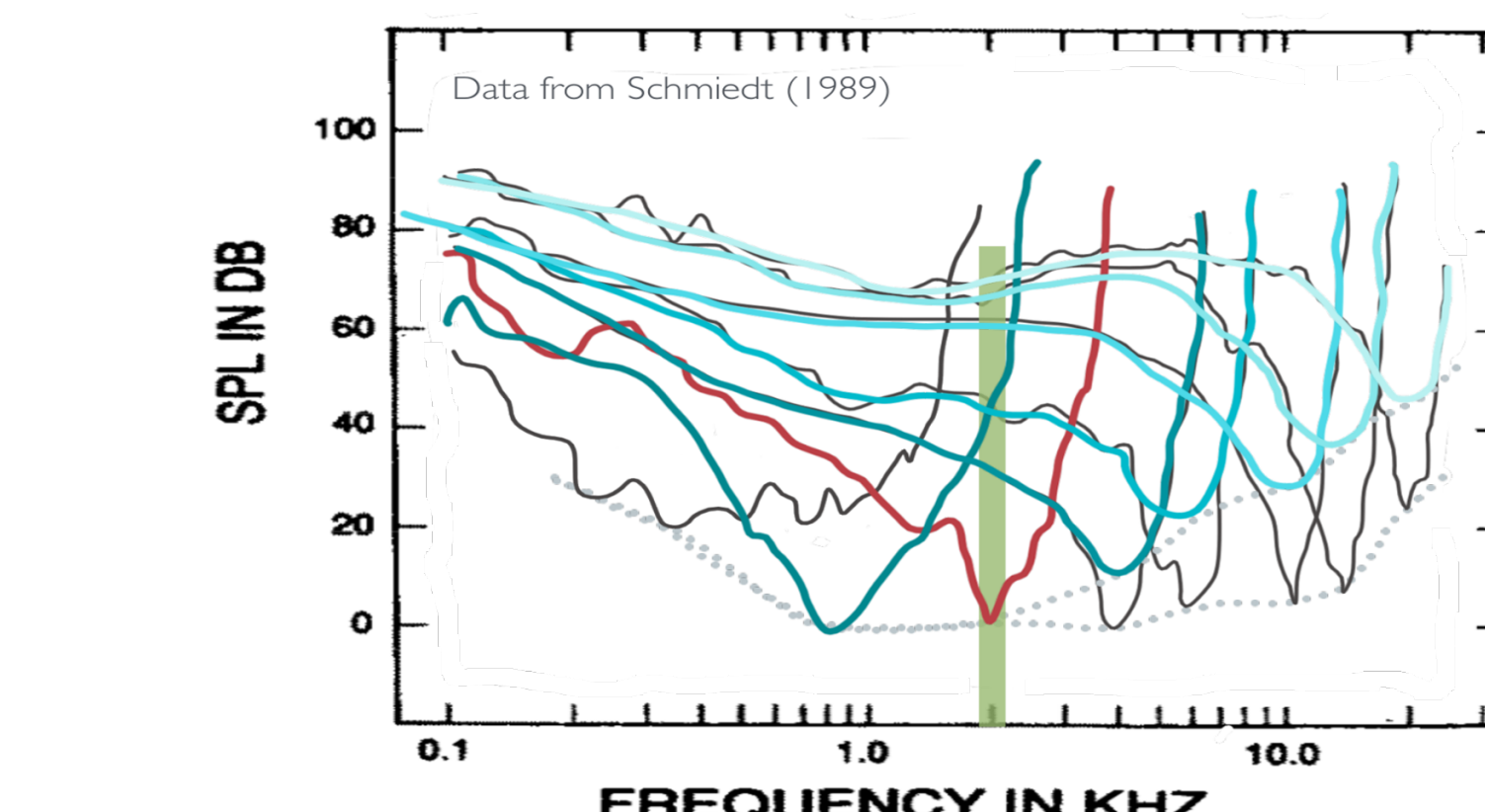


Fig.6 AN tuning curves stimulated with a high intensity SAM tone. Difference between NH and mild HI due to OHC dysfunction.

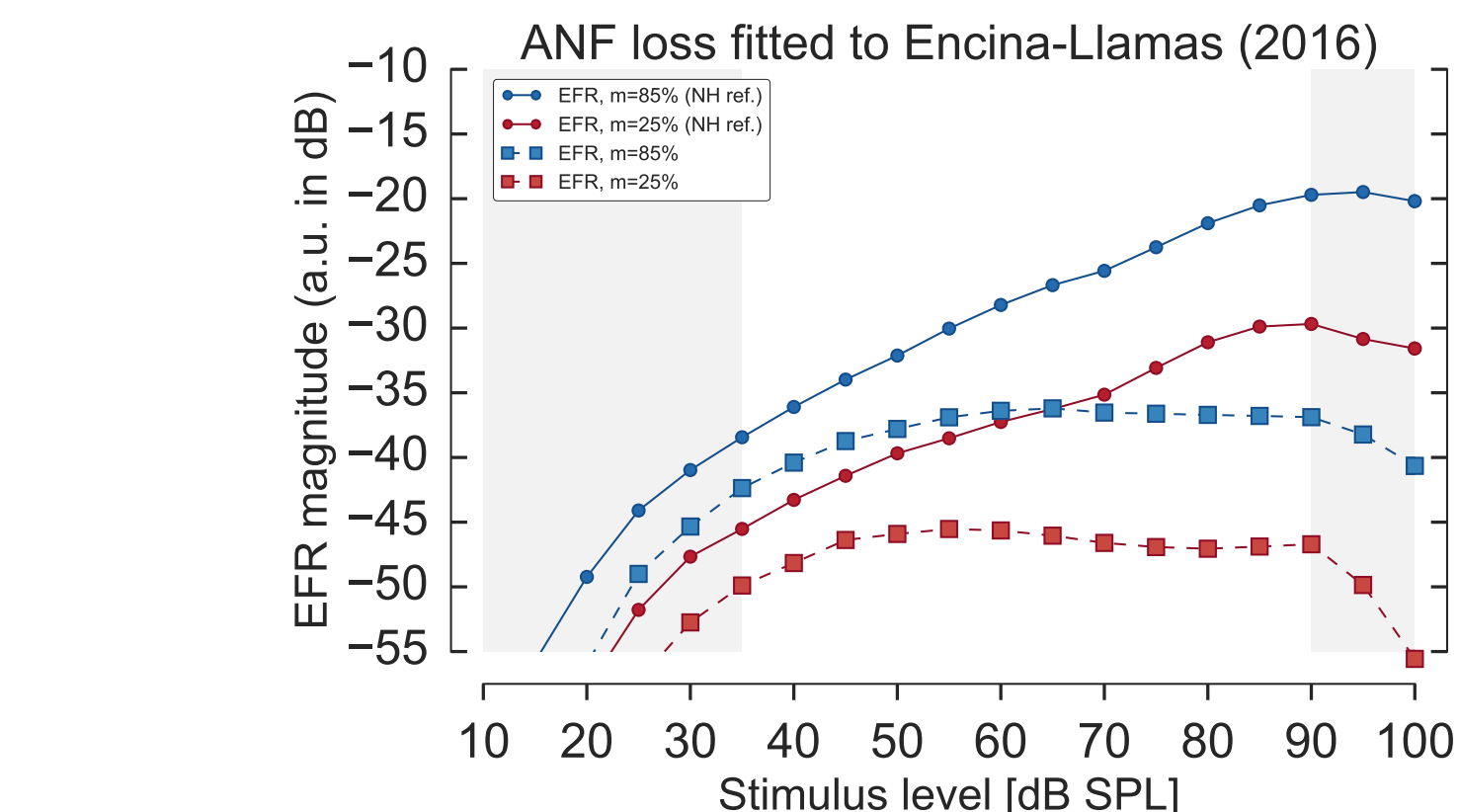


Fig.7 Simulated EFR level-growth functions to match the response from the mild-HI group in Encina-Llamas *et al.* (2016).

Simulations II

Broadband noise:

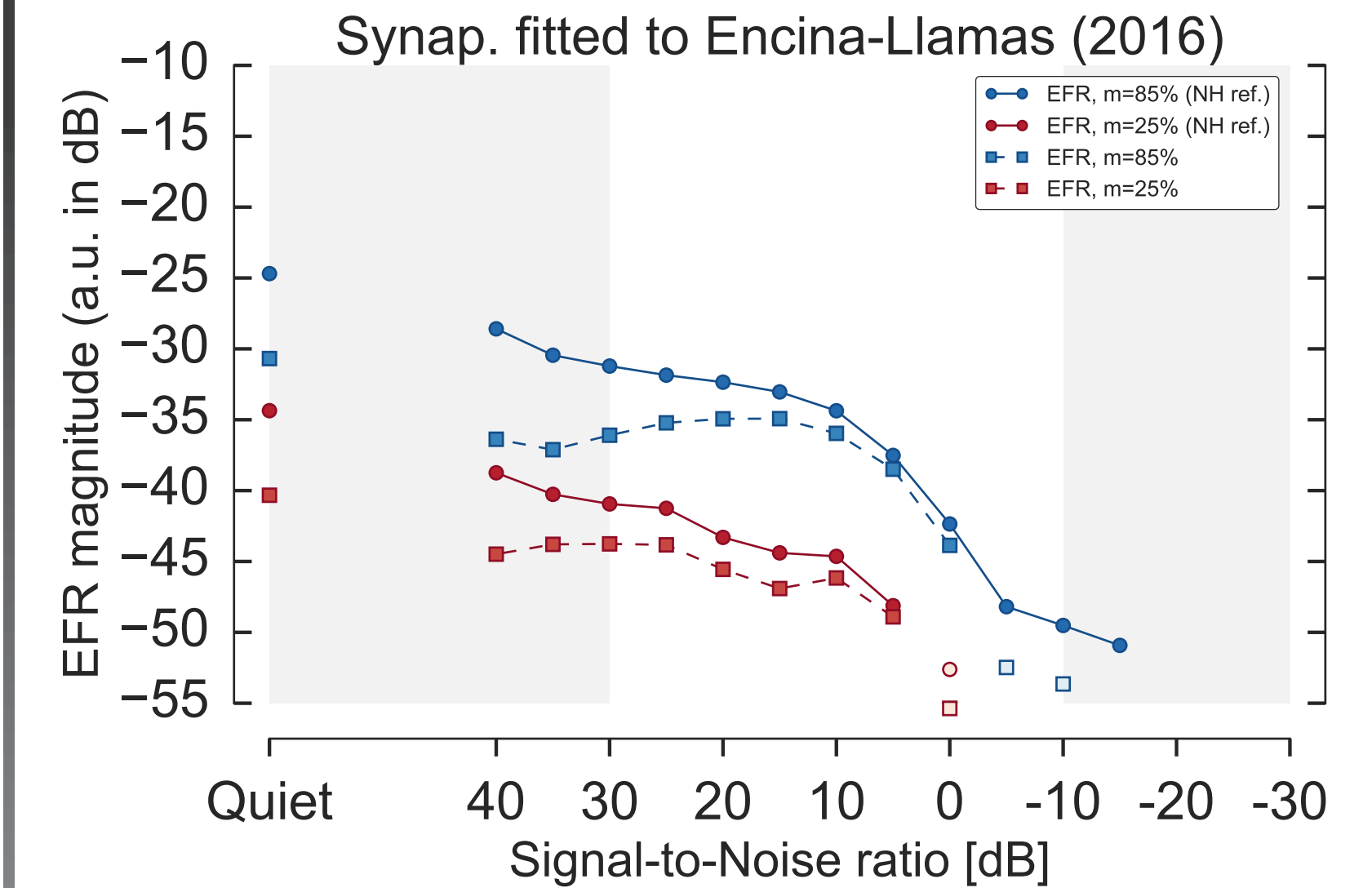


Fig.8 Simulated EFR in broadband noise. Comparison between the NH versus the synaptopathic simulation to match the NH? group in Encina-Llamas *et al.* (2016) as in Fig. 4.

Notch noise:

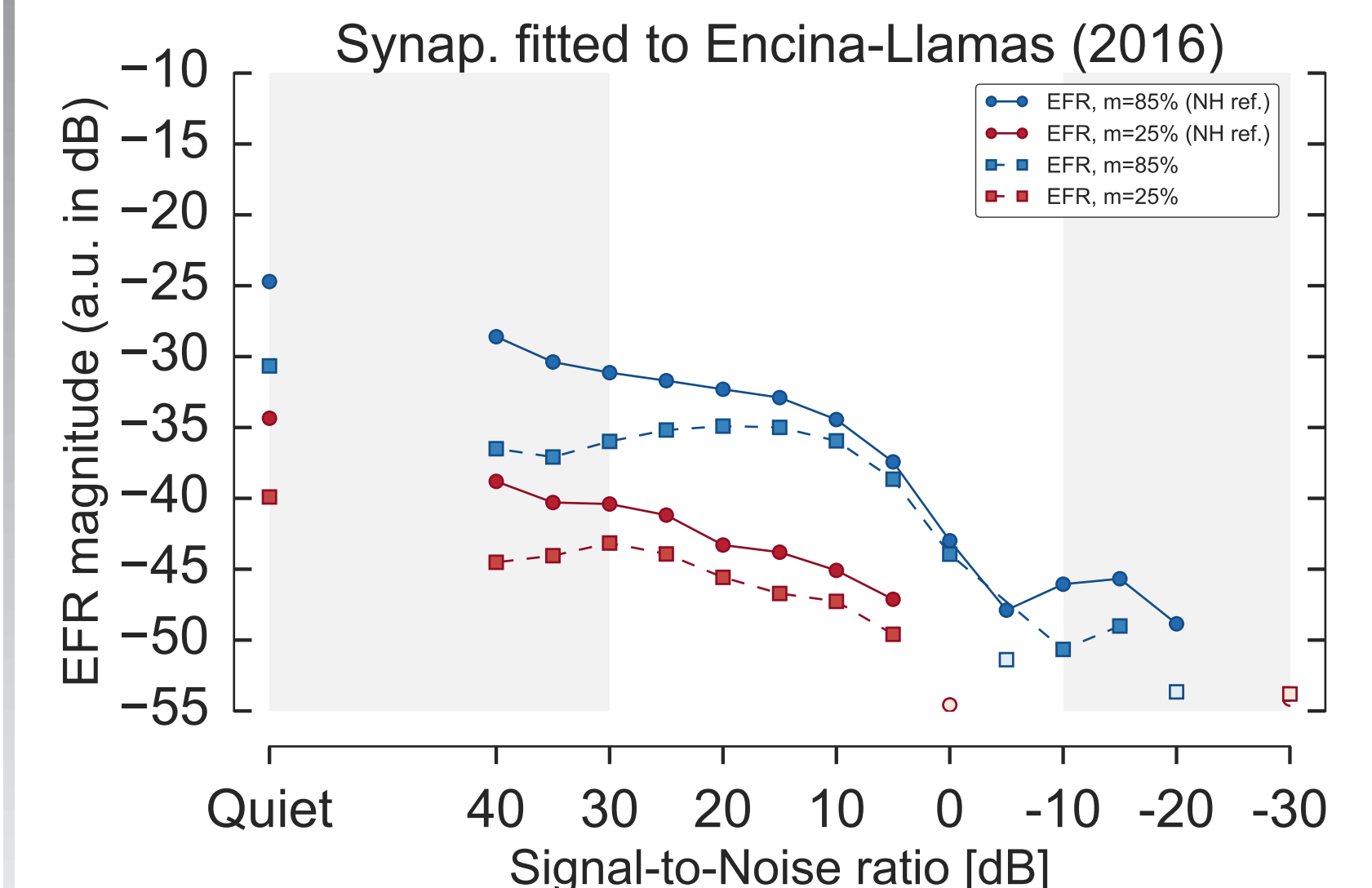


Fig.9 Simulated EFR in notch noise (800 Hz around f_c) as used in Bharadwaj *et al.* (2015). Comparison between the NH versus the synaptopathic simulation to match the NH? group in Encina-Llamas *et al.* (2016) as in Fig. 4.

Conclusion

- EFRs at high stimulus levels are dominated by the off-frequency contributions.
- EFRs are dominated by the responses from high-SR fibers.
- EFR level-growth functions from synaptopathic frequencies in exposed mice show similar trends to EFR functions in some NH human listeners (See poster PS 9 by Aravind Parthasarathy *et al.*).

ACKNOWLEDGMENT

Research supported by the Oticon Center of Excellence for Hearing and Speech Sciences (CHESS) at DTU in collaboration with CompNet at BU, and by DOD W81XWH-15-1-0103 (SGK) at HMS.

REFERENCES

- Bharadwaj *et al.* (2015). Individual Differences Reveal Correlates of Hidden Hearing Deficits. *J. Neurosci.* 35(5), 2161-2172.
- Encina-Llamas *et al.* (2016). Using auditory steady-state responses to evaluate auditory nerve integrity in normal-hearing and mild hearing-impaired listeners. *ARO MidWinter meeting*, 2016.
- Fernandez *et al.* (2015). Aging after noise exposure: acceleration of cochlear synaptopathy in "recovered" ears. *J. Neurosci.* 35(19), 7509-20.
- Kujawa and Liberman (2015). Synaptopathy in the noise-exposed and aging cochlea: Primary neural degeneration in acquired sensorineural hearing loss. *Hearing Res.* 330:191-199.
- Zilany *et al.* (2014). Updated parameters and expanded simulation options for a model of the auditory periphery. *J Acoust Soc Am*, 135(1), 283-6..